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Evaluation of Risks and Factors linked to Precocious Puberty

Review Article

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Abstract

Recent puberty-timing measurements have confirmed the continuation of accelerated sexual development know as precocious puberty [8, 4]. This shift in reproductive health has unknown health consequences. Current literature speculates that negative consequences of precocious puberty exist in a wide range from social angst to an increased risk of tumor development and cancers. Onset age of puberty is multifactorial, but there is strong evidence to suggest that an external factor may be impacting reproductive timing. Genetics, the environment, dietary choices, obesity, and estrogen disrupting compounds have all been purposed as potential causal agents. The rationale behind and practically of each of these potential determinants was evaluated to better understand and illustrate the connection to external sources and the problem of precocious puberty. Recognizing the presence and dangers of these different factors is critical in implementing methods and shifting behavioral patterns to decrease the risk of precocious puberty.

Introduction

Precocious puberty is diagnosed by breast tissue development; under arm and pubic hair growth; and menarche in females under the age of 8 and by genitalia enlargement; facial, under arm, and pubic hair growth in males under the age of 9 [13].

Since the 18 hundreds the onset age of menarche in females and other such measurements of sexual development have been measured as indicators of health. Zacharias and Wurtman [19] wrote in 1969, "there has been an acceleration of sexual development during the past 100 years". They quoted another author as concluding that, "'Age of menarche has been getting earlier by some 4 months per decade in Western Europe over the period of 1830-1960"' [19]. Although several factors were considered as causal agents most attributed the measurements as positive indicators of health and claimed the trend was a result of improved nutrition [8]. Even Zacharias and Wurtman [19] found studies that concluded, "that the economically privileged groups mature earlier than the under privileged, regardless of race or climate". The longevity of the trend is why certain groups are reluctant to believe that there is a sharp increase in the number of precocious puberty cases today. Even without ceding their position one is forced to reconcile the potentially devastating effects of even the continual gradual shift in sexual development.

An early onset age of puberty is observed in concordance with higher living standards and thus has been used in the past as an indicator of a population's health. "However, it is not a beneficial trait in either biological or social terms. Early age at puberty is associated with an increased risk of breast cancer, cardiovascular disorders and obesity" [18]. In 2007 Iwasaki et al., [9] published a study regarding age of puberty in Japanese girls and breast cancer later in life and concluded that menstrual and reproductive factors play "an important role in the development of breast cancer". Similarly Mucci et al., [14] linked individuals with hepatocellular carcinoma to a lower mean age at menarche in 2001. And Johansson et al., [10]. determined in 2005 that females "with menarche before age 11 (early) were more norm breaking, including being delinquents… women with early menarche were shorter and heavier. Had worse physical fitness and dieted more frequently". More likely to involve in sexual activity, more likely to act disobediently, and other social behavioral problems have all been seen as a short term issue of precocious puberty. The research clearly suggests that although previously used as a positive indicator of good nutrition and health early onset puberty is detrimental to the social and physical health of the individuals undergoing the changes, in both the short and long term.

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Precocious puberty is an obvious public health concern in that it causes both immediate and latent health issues. The onset age of puberty is multifactorial. With no one factor to target implementation programs have failed to combat the growing problem. It is thus important to determine which factors are on the causal continuum and have the potential to be controlled so as to decrease the occurrence of precocious puberty. Genetics plays a crucial role but it outside the scope of influence. Body Mass Index (BMI), dietary hormonal intake, and exposure to Endocrine Disrupting Compounds (EDC) will be evaluated as there is a potential to influence them.

Body Mass Index

An individual's BMI has been shown to be associated with precocious puberty [11]. When adjusting for socioeconomic groups it was found that, " the highest BMI value was always observed in girls with early age at menarche and the lowest in those with late age at menarche" [18]. Bouvattier and Piekowski's [5] publication on the latest findings regarding early puberty in 2014 concurred that BMI is of critical importance in puberty onset in both males and females. Pulling from a U.S. national survey [3]. in 2003 determined that, "Higher relative weight was strongly associated with increased likelihood of having reached menarche". The link between BMI and precocious puberty is abundant in literature. Why BMI plays such an important role is based on the hormonal changes associated with an increase in adipose tissue. Sorensen et al., in 2010 [17] linked several hormones to increased adiposity. These include insulin and leptin, which increase as the amount of adipose tissue increases, and testosterone and estrogen. An increase in adipose tissue was found to be linked with an increase in, "attenuated steroidogenesis in the testes as well as with increased aromatization of androgens to estrogens". These hormonal changes are all associated with precocious puberty. Although Sorensen et al., [17] conclude that BMI alone is not a sufficient explanation, it is apparent that as children develop adipose tissue their hormonal levels shift and the changes occurring have an impact on timing of puberty.

Drawing this connection is important because it adds even more importance to healthy childhood behavior and diet. Decreasing childhood obesity which has normally been associated with decreasing the risk of cardiovascular disease and diabetes can also now be associated with diminishing the risk of precocious puberty. Bouvattier and Piekowski [5] thus suggested that nutritional and behavioral changes may be a way to improve precocious puberty levels.

Dietary Intake of Hormones

There is a second piece to diet that must be addressed. The first as discussed is that excess calories, and diets high in sugar and fats can lead to increased BMI, hormonal changes, and thus an increase in one's risk of precocious puberty. The second aspect of diet is that direct consumption of certain products can result in hormonal changes and instigate precocious puberty. Hormones such as bovine growth hormone used to increase productivity of milk in cows has been under speculation as a potential culprit for the increase in the precocious puberty rates. Although Dr. Kaplowitz argues that the digestion of such a hormone is inconsequential and as stated the true link exists between BMI and precocious puberty [11]. A study by Lyytikainen et al., [12] in 2005 on the food consumption and daily caloric intake of females with early onset puberty supports this line of thinking. The study found that although the sample's average milk product consumption is high it is within the recommended amounts and not excessive like their saturated fats and carbohydrates. Soy based products have also taken heat as a potential delivery mechanism for hormones that could cause precocious puberty [2]. Disproved this thought and confirmed Lyytikainen findings on milk consumption in 2012 stating that crude analysis showed, "no difference in age at menarche between strata of… childhood vegetarian diet/soy consumption", and similar to Kaplowitz confirmed that, "Earlier age at menarche was observed among girls with high BMI". Although this does not disprove the possibility of influence of these factors it does suggest that their impact in minimal and not worth targeting as a public health official.

Estrogen Disrupting Compounds

Where concern does exist is with components such as bisphenol A (BPA) that can enter the body through foods and have the ability to disrupt normal hormone functions. BPA belongs to a family of chemicals classified as endocrine disrupting compounds (EDCs). "They are hypothesized to adversely affect female reproductive physiology by interfering with the organization of the hypothalamic-pituitary-gonadal axis" [1]. Commonly found in plastics, heating of food in microwaves using containers made of BPA has the potential for seepage into food and thus ingestion of the endocrine disruptor. Özen and Darcan reviewed the effects of EDCs on animals and humans in 2011 and concluded that time of exposure and dose are important factors but ultimately EDCs have been shown to cause multiple endocrine system dysfunctions as well as initiate pubertal development. Because a clear and evident link has been established between exposure to EDCs and precocious puberty it is important to evaluate the different EDCs and potential ways in which to reduce exposure [8, 15, 6, 16].

Roy et al., [16] published a comprehensive review of several man made EDCs. In regards to male precocious puberty the paper identified; dichlorodiphenyltrichloroethane (DDT), Dioxin, Bisphenol A, polychlorinated biphenyls (PCB), polybrominated biphenyls (PBB), Phthalate Esters, and Endosulfan as agents possible of influencing sexual development. For causal agents capable of influencing female sexual development the same paper identified; dichlorodiphenyldichloroethylene (DDE), Dioxin, Bisphenol A, PCB, PBB, Phthalate esters. These chemicals act in a variety of ways such as mimicking estrogen, blocking estrogen receptors, etc. but in all cases their presence has adverse effects on sexual development. Exposure to these chemicals must therefore be as limited as possible. Understanding the most frequent occurrence of said chemicals in the environment is thus important to public health officials attempting to decrease the rates of precocious puberty.

Discussion

Genetic factors obviously play a large role when discussing sexual development. Most of the studies evaluating BMI's link to precocious puberty had some element of control for race or ethnicity.

A genetic component is also heavily supported on its own in the literature [7] and [19]. However, seeing as how means to influence such a factor from a public health standpoint are limited it was not further discussed in this evaluation.

The link between BMI and precocious puberty is grossly apparent. Shifting diet and physical activity behavior in children may help to decrease the trend of increase precocious puberty cases throughout the world.

There are conflicting articles pointing to the harm and safety of products such as milk and soy. What was presented seems to be the most scientifically established, however, some EDCs such as Dioxin are most commonly exposed to humans through milk products. Thus although presumed safe a link may exist between consumption of said products and precocious puberty.

There is still much debate in literature as to which EDCs is most potent and at what dose and stage of development exposure is of highest risk. Some studies have failed to prove all of the EDCs mentioned are influential in altering sexual development. Study sizes and techniques vary for the testing of EDCs effects. Hamster trials as well as recall surveys done by children and parents have yielded a myriad of results. Further research is needed to accurately conclude the harmfulness of such compounds and their link to precocious puberty. Until then it seems viable to say that their presence is detrimental to the sexual development of children and should be avoided but the extent to which they may be influencing the trend of increase precocious puberty cases is unknown.

Conclusion

Regardless of one's belief as to whether the increase of precocious pubtery cases among males and females around the world is increasing at an alarmingly fast rate or simply following a trend that has persisted for hundreds of years there are obvious health implications of precocious puberty. These negative health outcomes should alarm public opinion and ensure that a new norm is not simply accepted. Scientific research has yet to identify one particular causal agent and thus implementation programs to fight precocious puberty have had no effective methodological approaches. However, as shown there are clear links between both BMI and EDCs on the sexual development of children. Thus in spite of the fact that precocious puberty is multifactorial and genetics and potentially other unmodifiable variable are in part to blame does not give public health officials the right to turn a blind eye to the problem. Efforts to decrease childhood obesity are acting in unison with beliefs that would better the problem of precocious puberty. More can be done to ensure EDCs are kept out of the environment and potential exposures to such products are limited in children. Such efforts have the potential to decrease the rates of precocious puberty thus decreasing risk of several negative health outcomes previously mentioned and increasing the general health status of the populous.

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